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TITLE OF CASE ***Do not include "a case report"***

Successful transcutaneous pacing following ventricular standstill during anaesthetic induction in a dog with third-degree atrioventricular block

SUMMARY ***Up to 150 words summarising the case presentation and outcome (this will be freely available online)***

Third-degree atrioventricular block is a haemodynamically unstable bradyarrhythmia frequently resulting in symptoms of lethargy, weakness and collapse. In this reported case, a four-year-four-month-old male neutered Cavalier King Charles spaniel diagnosed with third-degree atrioventricular block was referred for transvenous permanent pacemaker implantation. During induction of general anaesthesia the dog suffered cardiac arrest consistent with ventricular standstill, as indicated by cessation of ventricular electrical activity on the electrocardiogram monitor and the absence of a peripheral pulse. The prior placement of transthoracic pacing pads under sedation allowed for rapid commencement of temporary transcutaneous pacing and proved effective in achieving ventricular capture with re-establishment of cardiac output. The subsequent general anaesthesia for implantation of a permanent pacemaker was uneventful. This report considers the possible causes of ventricular escape rhythm suppression and highlights the importance of ensuring availability of a temporary pacing method from the outset when anaesthetising animals with unstable and symptomatic bradyarrhythmias.

BACKGROUND ***Why you think this case is important – why did you write it up?***

Permanent pacemaker implantation is indicated in dogs with symptomatic bradyarrhythmias including high-grade second degree and third-degree atrioventricular (AV) block, sick sinus syndrome (SSS) and sinus arrest (1). Cardiac arrest during implantation of permanent pacemakers is an occasionally reported complication of the surgical procedure itself, occurring consequently to ventricular fibrillation (2,3). Often, exacerbation of bradyarrhythmias may be detected following induction of general anaesthesia in these dogs, with one retrospective study identifying a requirement for temporary transcutaneous pacing (TCP) in 27 out of 77 cases (4). Although no set guidelines currently exist for activation of temporary pacing of anaesthetised dogs undergoing

pacemaker implantation, consistent initiation has been reported in incidences of asystole or with a heart rate of less than 20-25 beats per minute (bpm) (4). Prolonged periods of unstable and infrequent ventricular escape rhythms can result in myocardial ischemia which can increase the risk of ventricular fibrillation (2).

This case report documents an incidence of ventricular standstill on induction of general anaesthesia in a dog with third-degree AV block undergoing pacemaker implantation. When this occurred, the electrocardiogram (ECG) monitor showed cessation of the dog's ventricular escape rhythm, resulting in the absence of cardiac output. While the exact cause of ventricular standstill could not be determined, the potential for both sedative and anaesthetic agents to exacerbate inherent haemodynamic instability must be considered. In this case, TCP proved to be lifesaving and this report aims to highlight the importance of ensuring availability of a temporary pacing method prior to anaesthetising dogs with third-degree AV block.

CASE PRESENTATION ***Presenting features, clinical and environmental history***

A four-year-four-month old male neutered Cavalier King Charles Spaniel weighing 10.3 kg presented with a one-month history of lethargy and exercise intolerance, with two syncopal episodes during this period. On presentation, the dog had a heart rate of 32 bpm and strong synchronous pulses, with thoracic auscultation revealing a grade III/VI left systolic basal murmur. The dog had a regular respiratory pattern and a rate of 24 breaths per minute with no abnormalities detected on pulmonary auscultation. Mucous membranes were pink and moist with a capillary refill time of less than two seconds. Rectal temperature was 37.9 degrees Celsius.

Identification of a pronounced bradycardia indicated a requirement for ECG analysis, which provided a diagnosis of third-degree AV block with an atrial rate of 144 bpm and ventricular escape rate of 36 bpm. Echocardiography revealed mild thickening of the mitral valve leaflets with regurgitation and visualisation of global cardiac dilation suspected to be secondary to chronic bradycardia. A mildly elevated platelet count of 528x10^9/L was the only abnormality on haematology and biochemistry results were all within reference range. The decision was taken to anaesthetise the dog for permanent transvenous pacemaker implantation.

Following placement of a 22G intravenous catheter in the left cephalic vein, the dog was premedicated with 20 µg/kg acepromazine (Acecare 2 mg/ml; Animalcare, UK) and 20 µg/kg buprenorphine (Buprecare 0.3 mg/ml; Animalcare, UK) intravenously. After approximately 25 minutes, sedation was adequate to enable clipping of both hemithoraces and the bilateral placement of paediatric pacing electrodes (EDGE System radiotransparent electrodes with QUIK-COMBO connector, Physio-Control) between intercostal spaces 5-7 at the level of the heart apex, secured in place using non adhesive elasticated bandaging. Pacing leads from the transcutaneous pacing system (Lifepak-20; Medtronic, USA) were attached to the paediatric pacing electrodes and ECG leads were connected to ECG pads placed on the paw pads of the left and right forelimb and left hindlimb. Placement of a Doppler ultrasound probe over the left dorsal metatarsal arterial pulse allowed for continuous audible monitoring of the peripheral pulse and intermittent non-invasive arterial blood pressure measurements.

The dog was moved into theatre and general anaesthesia was induced with a total dose of 3.3 mg/kg propofol (PropoFlo Plus 10 mg/ml; Zoetis, UK) intravenously administered to effect with the dog positioned in left lateral recumbency. The trachea was intubated with a cuffed 6.5 mm endotracheal tube (ETT). The ETT was connected to a circle breathing system and general anaesthesia maintained with sevoflurane vaporised in oxygen. Immediately following the period of general anaesthetic induction and tracheal intubation, the right femoral arterial pulse, which was being continuously monitored, became undetectable with simultaneous loss of the previously audible Doppler signal. On observation of the ECG trace it was noted that the third-degree AV block had converted to ventricular standstill, with only P waves visualised and complete loss of ventricular escape complexes. While P wave rate was not recorded, their frequency on the ECG

monitor was observed to decline in comparison to preinduction values. Although the animal maintained spontaneous respiration, the end tidal carbon dioxide values were observed to decrease on the capnograph trace from approximately 40 mmHg (5.4 kPa) to values < 30 mmHg (4.0 kPa), suggesting a sudden drop in cardiac output.

INVESTIGATIONS ***If relevant***

DIFFERENTIAL DIAGNOSIS ***If relevant***

Absence of a peripheral pulse and cessation of ventricular escape complexes on the ECG resulted in a diagnosis of ventricular standstill.

TREATMENT ***If relevant***

Following detection of ventricular standstill, TCP was immediately initiated at a rate of 80 bpm. It is approximated that the time between loss of a peripheral pulse and commencement of TCP was no greater than 40 seconds in duration. The TCP current was incrementally increased until capture was observed, as indicated by the appearance of a wide QRS complex proceeding each pacing spike on the ECG monitor and the return of a palpable peripheral arterial pulse. As a result of temporary TCP causing generalised extraneous muscle contraction, 0.1 mg/kg atracurium (Atracurium besilate 10 mg/ml; Hameln Pharmaceuticals, UK) was administered intravenously to achieve neuromuscular blockade and prevent the associated movements. Volume controlled intermittent positive pressure ventilation (IPPV) was immediately started at a tidal volume of 110 ml and respiratory rate of 12 breaths per minute.

OUTCOME AND FOLLOW-UP

General anaesthesia for implantation of the transvenous permanent pacemaker lasted for 150 minutes in duration and was uneventful. Following exteriorisation of the right jugular vein, an incision into the vessel enabled introduction of a passive fixation permanent pacing lead into the apex of the right ventricle under fluoroscopic guidance. Whilst occasional ventricular premature complexes were observed on the ECG during presence of the permanent lead in the right ventricle, non-invasive mean arterial pressures were consistently maintained above 60 mmHg throughout general anaesthesia.

Following endocardial lead placement, TCP was discontinued, resulting in heart rate dropping to 30 bpm and recurrence of the dog's intrinsic third-degree AV block. The permanent pacing lead was subsequently attached to a pulse generator set at 70 bpm and 3.5 volts to achieve capture. Following securement of the permanent pacing lead to the jugular vein, which was ligated cranially, a subcutaneous pocket was created in the right neck region for placement of the pulse generator. Transient detachment of the pacing lead from the generator allowed for its tunnelling under the skin and then subsequent reconnection. The pulse generator was placed within the subcutaneous pocket, with subcutaneous tissue and skin closure achieved with 2 metric poliglecaprone 25 (Monocryl; Ethicon) and 2 metric polydioxanone (PDS II; Ethilon) respectively.

Recovery from general anaesthesia was smooth and the dog was taken to recover in the intensive care unit. In order to ensure the dog remained calm and to prevent the risk of pacing lead dislodgement, 20 µg/kg acepromazine was administered intravenously every 6 hours overnight. In addition, avoidance of neck lead usage and prevention of any blood sampling from the jugular veins was instructed. Although the surgery was deemed to be minimally invasive, 20 µg/kg buprenorphine was administered intravenously every 6 hours overnight for the purpose of analgesia provision. The dog was discharged from the hospital four days post operatively, with the period of rest during hospitalisation anticipated to allow for initial stabilisation of the pacing lead. During the period of hospitalisation, no additional complications were reported.

DISCUSSION ***Include a very brief review of similar published cases***

Third-degree AV block is thought to most commonly occur as a result of a structural lesion within the cardiac conduction system but may also arise due to functional causes such as hyperkalemia or intoxication with calcium channel blockers (5,6,7). In a case series of four dogs with concurrent mitral valve endocardiosis, third-degree AV block was suggested to occur as a result of the altered haemodynamics causing mechanical damage to the myocardium and consequential fibrotic lesions developing within the AV conduction system (6). Third-degree AV block occurs with disturbance of conduction between the atria and ventricles, characterised on the ECG by P waves that are unassociated with QRS complexes. While the pacemaker cells of the sinoatrial node continue to depolarise and cause atrial contraction at a normal intrinsic rate, the absence of AV conduction results in development of a ventricular escape rhythm. Escape rhythms

originate due to the presence of slowly depolarizing subsidiary pacemaker cells within either the AV junction or the ventricles and are characterised by widened QRS complexes occurring independently of P wave activity (8). Symptoms of third-degree AV block occur as a result of reduction in cardiac output, a product of heart rate and stroke volume, and typically include lethargy, weakness and collapse (9). In consideration that many dogs with third-degree AV block are at high risk of sudden death, permanent pacemaker implantation is the treatment of choice in cases where the cause is idiopathic or untreatable (9).

In this reported case, the dog presented outside of working hours with a symptomatic bradycardia and transthoracic pacing pads were placed as a precaution in case of haemodynamic instability under general anaesthesia. One retrospective study by Ward et al. (10) reviewing the medical records of 97 dogs undergoing permanent transvenous pacemaker implantation identified significantly more major complications with procedures occurring out of hours, although this was attributed to differences in personnel factors rather than provision of temporary pacing. While the use of TCP in dogs undergoing pacemaker implantation has previously been associated with reductions in long term survival, those requiring external pacing may represent the proportion of a population initially presenting with more severe disease and greater haemodynamic instability (11).

Placement of transthoracic pacing pads to the hemi thoraces was in this case facilitated following sedation of the dog with buprenorphine and acepromazine, the latter of which was selected for its anxiolytic effect and long duration of action. The vasodilatory effects of acepromazine can additionally be beneficial in reducing afterload in cases of degenerative mitral valve disease, a comorbidity frequently identified in dogs requiring permanent pacemaker implantation (12, 13). Buprenorphine, a partial mu agonist opioid, was selected preferentially in this reported case over a full mu agonist opioid in an attempt to maintain heart rate and because permanent pacemaker implantation via a transvenous approach was deemed to be minimally invasive surgery (14, 15). Alternative opioids which could be considered are butorphanol or pethidine. Unlike other full mu agonist opioids, pethidine has anticholinergic effects that maintain heart rate (16). Buprenorphine's longer duration of action compared to butorphanol and pethidine provided the advantage of enhancing the sedative effects of acepromazine, while also ensuring analgesia provision for the duration of surgery and negating the requirement for repeated administrations intraoperatively.

Detection of ventricular standstill following anaesthetic induction prompted immediate commencement of TCP. While it is difficult to ascertain the cause of ventricular escape rhythm cessation, the potential contributing effects of both the premedicant agents and propofol must be considered in this case. Previously, acepromazine has been identified to provide some protection against ventricular arrhythmias in halothane anaesthetised dogs receiving epinephrine infusions (17, 18). The anti-arrhythmic properties of acepromazine have been attributed to a number of possible mechanisms, including stabilisation of the resting membrane potential in cardiac myocytes (17, 19) and antagonism of alpha 1 adrenoreceptors within the myocardium (18). Acepromazine can additionally cause a vagally meditated bradycardia, although typically at doses higher than those used in clinical practice (20). Alternatively, buprenorphine administration can result in a vagally mediated bradycardia, with acetylcholine stimulating reductions in the rate of spontaneous depolarisation of the sinoatrial node and subsidiary pacemaker cells (12).

In consideration that ventricular standstill was identified following anaesthetic induction, it is possible that propofol exerted a suppressive effect on ventricular depolarisation. Propofol can cause bradycardia, albeit via a different mechanism to that of acepromazine and buprenorphine, with suppression of the funny current responsible for the initial depolarization of pacemaker cells being one proposed mechanism of action (21). Alternative options for induction of general anaesthesia in this case could have included etomidate or ketamine, in combination with a benzodiazepine. Etomidate is considered to be the anaesthetic induction agent with the least

cardiovascular depressant effects, although it can induce vomiting, excitation and myoclonus (22). The use of ketamine has been described in the anaesthetic management of dogs undergoing pacemaker implantation (23). Ketamine has sympathomimetic effects but it can also have direct negative inotropic effects, particularly in patients with catecholamine depletion (24). Administration of propofol in this case may have exacerbated the pre-existing haemodynamic instability in this dog and contributed to ventricular standstill.

The requirement to initiate TCP in dogs with third-degree AV block following detection of asystole or profound bradycardia during pacemaker implantation is not uncommon (4,11). The Reassessment Campaign on Veterinary Resuscitation (RECOVER) guidelines recommend basic life support (BLS), consisting of airway management, manual ventilation and chest compressions, following detection of asystole (25). When arrest is suspected to be of primary cardiac origin, restoration of circulation should be prioritised in order to maintain coronary and cerebral perfusion (25). In comparison to external chest compressions, which passively expel blood from the heart, external pacing methods result in electromechanical capture of the ventricles and have been shown to result in increased cardiac output (26,27). In human medicine, pacing is recommended on detection of asystole if the patient is known to have high-grade second or third-degree AV block and when P waves are still visualised on the ECG (28). The practicality of preparing and initiating temporary pacing has been acknowledged as a possible cause of delay in restoring circulation and should be considered (29). In this case, placement of pacing pads prior to anaesthetic induction enabled pacing to commence almost immediately following loss of a ventricular escape rhythm and demonstrates the importance of having a temporary pacing modality when anaesthetising dogs with third-degree AV block.

In this case, TCP was effective in restoring cardiac output and administration of positive chronotropic drugs as part of advanced life support (ALS) was not considered necessary. One retrospective study of 77 dogs presenting for pacemaker implantation identified that only 5% were observed to be atropine responsive on initial presentation, demonstrating the unpredictable effects of anticholinergics in situations of cardiac conduction system disturbances (10). Despite the low percentage of atropine responsive dogs, Ward et al (10) reported pre-emptive administration of anticholinergics to all dogs prior to anaesthesia for pacemaker implantation, in efforts to avoid vagally mediated reductions in ventricular response rate associated with opioid administration and general anaesthesia. Interestingly, depending on the location of subsidiary pacemaker cells, atropine may result in an increase in the rate of ventricular escape rhythms (9). When anaesthetising dogs with third-degree AV block, atropine should not be relied upon to avoid cardiac arrest; An appropriate and effective means of temporary pacing must be in place.

First introduced for humans in the 1950's, TCP provided a non-invasive alternative to temporary transvenous pacing (TVP) and hence removed the risks of haemorrhage, infection and arrhythmias occasionally reported with the latter (30, 31, 32). The predominant difficulty surrounding the use of temporary TVP in clinical practice however is the time and expertise required for placement; the temporary pacing lead must be guided into the right ventricle via a saphenous or femoral vein, the practicalities of which may be challenging in smaller patients (33). Placement of a temporary TVP lead into the right ventricle is enhanced by visualisation of the pacing lead under fluoroscopic guidance but this imaging technique may not always be readily available and increases radiation exposure to personnel. Provided that the pacing pads are placed correctly and a suitable current and pacing rate is applied, TCP offers a quickly accessible alternative to TVP and may be a more appropriate option in emergency situations when time is critical.

A requirement for TCP to treat asystole or profound bradycardia is commonplace in dogs undergoing general anaesthesia for permanent pacemaker implantation (4, 23), demonstrating the importance of access to a temporary pacing method in symptomatic bradyarrhythmic animals.

Effective approaches to TCP have previously been well described in the veterinary literature (4, 30). The predominant disadvantage of TCP when compared to TVP is the requirement for greater currents in order to achieve ventricular capture, which can result in significant extraneous muscle contractions. Muscle contractions caused by TCP are reported to be painful, necessitating appropriate analgesic provision. When such pacing is performed on an elective rather than emergent basis, it should not be done so in the conscious animal. Techniques such as positioning the negative anode on the left hemithorax and ensuring electrodes are positioned at the level of the heart apex may assist in achieving the lowest effective current for capture, but any movement under general anaesthesia is undesirable for surgery (15, 34). The skeletal muscle contractions induced from TCP under general anaesthesia can result in inability to provide effective IPPV and can therefore risk hypoxaemia (35). Administration of neuromuscular blocking agents e.g. atracurium, can abolish TCP induced skeletal muscle contraction but will consequently result in a requirement for IPPV provision due to concurrent blockade of the respiratory muscles. Administration of neuromuscular blocking agents requires careful monitoring, ideally with the use of a peripheral nerve stimulator, as postoperative residual block in recovery can result in upper respiratory obstruction and impaired ventilation (36, 37).

This reported case demonstrates the importance of having a method to instigate temporary cardiac pacing available prior to anaesthetising animals with unstable and symptomatic bradyarrhythmias. Dogs with third-degree AV block are haemodynamically unstable and the cardiovascular effects of both sedative and anaesthetic agents should be considered prior to their administration. The value of ongoing monitoring in both the sedated and anaesthetised animal should not be underestimated and the use of Doppler and regular peripheral pulse palpation will greatly facilitate the rapid detection of asystole. TCP provides a non-invasive and rapid approach to treating asystole in animals with third-degree AV block, although an understanding of temporary pacemaker settings and neuromuscular blocking agents is additionally necessary for its effective and safe use.

LEARNING POINTS/TAKE HOME MESSAGES ***3 to 5 bullet points – this is a required field***

Availability of a method of temporary cardiac pacing and familiarity with its use is critical when anaesthetising any dog with a bradyarrhythmia, particularly in unstable and symptomatic cases.

The cardiac depressant effects of premedicant and anaesthetic induction agents may exacerbate pre-existing haemodynamic instability in dogs with third-degree AV block.

Induction of general anaesthesia in bradyarrhythmic dogs warrants continuous monitoring of a peripheral pulse in order to quickly recognise and treat asystole should it occur.

Effective and safe use of temporary transcutaneous pacing requires appropriate positioning of electrode pads and application of suitable pacing settings to achieve capture.

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